Grand Rounds in Environmental Medicine: Information on MCS Needed

In the "Preface" to *Grand Rounds in Environmental Medicine: Cases from an Emerging Discipline*, Howard Hu (2003) stated that

> Part of the value of Grand Rounds stems from the appreciation that in real life, patients rarely present with cut-and-dry cases of illnesses as they are described in medical school. Real people have unique combinations of preexisting health conditions, symptoms, and complaints.

No truer words have been spoken about a chemically injured patient—but more words could be added. Alas, mainstream doctors are not trained adequately to diagnose symptoms and adverse health events from pesticide poisoning, not even when the patient describes actual exposures to the doctor. Try to imagine what it is like for a patient suffering from perfume poisoning. I certainly hope that Grand Rounds in Environmental Medicine will do much to change that.

I was pleased to see the list of environmental exposures that affect human health—both those that are more common, such as lead, mercury, asbestos, organophosphate pesticides, and polychlorinated biphenyls, and others that are more unusual, such as manganese, bromine, and carbon disulfide (Hu 2003). As Hu (2003) also noted, other factors are beginning to be accepted as environmental health hazards, such as cockroach allergen and violence.

Hu (2003) continues:

> Some [Grand Rounds cases] pertain to illnesses arising from occupations that entail combinations of exposures that may have acted synergistically. Some arise out of new research on illnesses and exposures that had not previously been linked together, such as infant pulmonary hemorrhage/*Stachybotrys* mold and possible estuary-associated syndrome. Others explore illnesses that are still of uncertain etiology and biology, such as multiple chemical sensitivities.

I would be even more pleased if modern flavors and fragrances appeared in the list.

I am often displeased to see articles in mainstream newspapers in which doctors advise patients with asthma to rid their homes of the "usual suspects"—cats, cockroaches, and dust and dust mites, sometimes including mouse feces and mold for good measure—without mentioning the potential harm that fragrance chemicals can cause for people with asthma or other diseases exacerbated by fragrance products.

Fragrances are not benign. Each scent is composed of untold combinations of highly volatile petrochemical derivatives. How many of these chemicals are in the air, being breathed and absorbed by user and nonuser alike? People have good reasons to avoid using scented products; for example, an individual may have adverse reactions to these products, or a pregnant woman may be concerned about the welfare of her fetus. Illnesses that may be affected by petrochemically derived flavors and fragrances include asthma and other respiratory diseases, migraines and other headaches, neurologic events, and even cancer.

Unfortunately, people who do not use scented products become users nonetheless—from sharing space with people who use these products. These chemicals cross not only the blood–brain barrier but also the blood–placental barrier. They go directly through the skin to target organs and are stored in adipose tissue. Whether these toxic chemical mixtures are used as flavors or fragrances, they play a major role in the adverse health events experienced by millions of men, women, and children, as well as developing fetuses. Too often, mainstream doctors do not consider that modern pharmaceuticals may be petrochemically derived and could contain petrochemically derived flavors and fragrances. No wonder there are skyrocketing rates of asthma and cancers, as well as high rates of iatrogenic diseases and deaths.

The public needs to learn of this information through sources such as *EHP*. Although it is possible to dig deeply enough to find some information on government agency websites, the general public cannot readily and easily access the information from the Food and Drug Administration, the U.S. Environmental Protection Agency, the Consumer Products Safety Commission, the Agency for Toxic Substances and Disease Registry, or the Centers for Disease Control and Prevention. These agencies have not done all they could to protect public health and well-being regarding adverse health events associated with synthesized flavors and fragrances.

As *EHP* builds a "compendium of Grand Rounds in Environmental Medicine," I fervently hope that information will be included about adverse health effects caused, triggered, or exacerbated by modern flavors and fragrances, particularly those of the last 30 years. The tens to hundreds of chemicals used to build each scent can cause the varieties of diseases and illnesses associated with multiple chemical sensitivity (MCS).

It is past time for public agencies, mainstream medical doctors, and everyone touched in any way by this life-threatening and life-changing malady to learn more about the effects of petrochemically derived flavors and fragrances. We all are stakeholders when it comes to breathing.

I appreciate *EHP* for providing the forum for presenting and discussing important topics such as this one. Additional information on MCS is available on the web (Environmental Health Network 2004; Fragranced Products Information Network 2004; Health Care Without Harm 2004; Manura 1998).

The author declares she has no competing financial interests.

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The Simple Truth about Multiple Chemical Sensitivity

Renee Twombly's news article "The Simple Truth about MCS" (Twombly 2003) ignores the plausible physiologic mechanism (described in the same issue of *EHP*) that answers each of the major questions about multiple chemical sensitivity (MCS) (Pall 2003). Given the many puzzling features of MCS and the previous claims that there cannot be a plausible physiologic mechanism to explain it, the finding of a physiologic mechanism (Pall 2002, 2003; Pall and Satterlee 2001) is a landmark in our understanding of this illness.

This mechanism provides answers to each of the most important and previously intractible questions about MCS (Pall 2003), such as:

- How can the reported exquisite (approximately 1,000-fold) sensitivity to chemicals be produced?
- How are the two main classes of chemicals—acetylcholinesterase inhibitors (organophosphates and carbamates) and hydrophobic organic chemicals—involved?
- How can previous chemical exposure induce such sensitivity?
- Why is MCS chronic?
- How are the characteristic symptoms of MCS generated?

- Why does MCS have multiple overlaps with chronic fatigue syndrome and fibromyalgia?
- How might neurogenic inflammation be generated as part of the MCS mechanism?
- How can lowering of several steps in porphyrin synthesis occur as a consequence of the MCS mechanism? (Classical porphyria is caused by lowering a single step in the pathway, whereas in MCS, several steps in the pathway are low.)

We look to articles such as Twombly's to connect the most important dots and produce for the reader the essence of the resultant pattern. I hope that Twombly's failure to do so for MCS will be a temporary lapse.

The author declares he has no competing financial interests.

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Multiple Chemical Sensitivity: Response to Pall

Renée Twombly's news article (Twombly 2003) was not intended to be a comprehensive discussion of multiple chemical sensitivity. Rather, as per the standing format of the Science Selections portion of *EHP*, Twombly was assigned to summarize the findings of a particular research article in the same issue (Gibson 2003). It is beyond the scope of Science Selections articles to "connect the most important dots" of whatever research topic they summarize, and Twombly's failure to do so in her article in no way reflects upon her.

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Accuracy of Declared Conflicts of Interest

Growing evidence shows that the findings and conclusions of researchers with financial conflicts of interest are significantly more likely to favor the interests of their for-profit supporters (Bekelman et al. 2003; Swaen and Meijers 1988), as does evidence of the takeover of the institutions of science by for-profit corporations (Blumenthal et al. 1996; Willman 2003). However, many authors remain reluctant to declare the financial benefits they receive (Krimsky and Rothenberg 2001). In elucidating its new conflict of interest policy, the *British Medical Journal* theorized:

> We have two hypotheses to explain this. Firstly, authors think that an admission of a conflict of interest implies wickedness. We don't think so. Secondly, authors are confident that they have not been influenced by a conflict of interest and so don't tell us they have one. Our response is that bias works in subtle ways and that none of us is blessed with knowledge of our own motivations and mental mechanisms. (Smith 1998)

EHP too has strengthened its conflict of interest policy—to now require authors to provide a public declaration of competing interests that could constitute a real, potential, or apparent financial conflict, and require they certify their freedom from competing interests to conduct all aspects of research (*Environmental Health Perspectives* 2003).

Unfortunately, author reluctance is evident. For example, Starr (2003) (of TBS Associates) recently published a worthy commentary in *EHP* in which he analyzed the controversial and high-financial-stakes question of the cancer potency of dioxins; yet he states,

> Partial support for this work was provided by the Chlorine Chemistry Council [CCC]. The author declares he has no conflict of interest.

In the same issue, Gibson et al. (2003) reported on their investigation of the efficacy of therapies for multiple chemical sensitivity (MCS), as perceived by those taking them. They state,

> This research was supported in part by a grant from the Chemical Injury Information Network [CIIN] and a James Madison University Faculty Summer Research grant. The authors declare they have no conflict of interest.

EHP's long-standing transparency of author affiliation and funding allows readers to judge both claims. The corporations that make up the CCC derive a huge financial benefit in seeing dioxins and other chlorinated chemicals declared safe to the biosphere. However, the CIIN, an influential support group for those with MCS, gains a small financial benefit from seeing MCS declared a prevalent hazard, even if the benefit is large relative to the CIIN's small size. Readers can factor in the CCC's dedication to the narrow interests of for-profit corporations (almost entirely financial gain) and the CIIN's bias toward the very public interest of health. University and government funders such as the National Institutes of Health also represent the broad public interest and are biased toward health issues.

Journal editors should require declarations (including for correspondence) to be more truthful. Editors should also declare their financial conflicts.

What about refusals to declare a financial conflict when the funding source does not indicate its presence? Journals should regularly publish a reminder about the importance of declaring all conflicts of interest, and they should solicit readers for notification of undisclosed conflicts and publish any that are received and verified.

Author bias is inevitable, but it contributes to scientific discourse—the only way for humanity to gain knowledge (e.g., when a journal publishes, without a conflict of interest declaration, the proceedings of symposia that include industry-affiliated authors). Full transparency is critical to the advancement of knowledge.

The author declares he has a competing financial interest because he received payment from a public-interest nonprofit agency (Women's Voices for the Earth) and a trial attorney, both of whom could profit from the service he provided. He did not receive funding for this letter.

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Conflicts of Interest: Gibson's Response

I applaud Tweedale's effort to ferret out and expose true conflicts of interest in regard to health research. However, he is barking up the wrong tree when he suggests that conflict of interest was present in my study of perceived treatment efficacy for therapies for multiple chemical sensitivity (MCS) (Gibson et al. 2003). Although I admit to having a strong interest in and caring deeply about issues relating to my topic of research, I do not think that this distinguishes me from any other researcher.

My funding was from the Chemical Injury Information Network (CIIN), a nonprofit organization that works to provide education and advocacy regarding MCS. Neither the CIIN nor I have any vested financial interest whatsoever in any of the treatments researched. My purpose for the study was to examine resource allocation for and efficacy of treatments for a currently delegitimized condition. The CIIN has an interest in gathering and providing information to those with MCS about this issue, but the organization neither advocates nor benefits from the use or sale of any particular treatment. Tweedale's suggestion that the CIIN may gain financially from "seeing MCS declared a prevalent hazard" seems inappropriate, given the study's focus on treatment and not prevalence.

Finally, I clearly disagree with Tweedale's suggestion that even funding from the National Institutes of Health (NIH) is suspect because the NIH has an interest in health. Nonprofit organizations have been funding health research for decades and, in my mind, having an "interest in health" is in no way synonymous with a financial vested interest.

The author declares she has no competing financial interests.

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Editors' note: *Because the phrase "conflict of interests" can be interpreted so broadly that almost everyone could be considered to have a conflict, we modified our policy for declaring conflicts of interest in December 2003 (Environ Health Perspect 111:A900–905).* EHP *now requires authors to declare "competing financial interests." The new policy is available on the web in our updated Instructions to Authors (**http://ehp.niehs.nih.gov/docs/admin/edpolicy.html**); the new downloadable form is also available (**http://ehp.niehs.nih.gov/cfi.pdf**).*

Because EHP *is not in the position to confirm the accuracy of disclosure statements made by our authors, we hold authors responsible for providing accurate information.* EHP *authors can expect scrutiny of their statements by our readers and by the authors' own employers. We welcome letters to the editor that address alleged inaccuracies of declarations of competing financial interests.*

Six Modern Plagues

In his generally positive review of my book, *Six Modern Plagues and How We Are Causing Them* (Walters 2003), Donald S. Burke (2004) neglected to mention that *Six Modern Plagues* goes out of its way to differentiate between fact and theory. I stated, for example, that the basic mechanism of the emergence of human immunodeficiency virus "is still unproven," that "there is some evidence" for *Salmonella* drug resistance being acquired from fish farms in Asia, that the widely accepted belief that mad cow disease originated from scrapie in sheep is "still just a hypothesis," and that "perhaps" an infected person first introduced West Nile virus into the United States. However, Burke's blanket assertion in the review that "an infected arriving human could not have been the origin of the West Nile epidemic in Queens, New York" confuses fact with theory, indeed. This may be his informed opinion, but it is far from scientifically established fact.

The author receives royalties fom the publication and distribution of this book.

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Hypothyroxinemia, Iodine Deficiency, and Subtle Changes in Migration and Cytoarchitecture

In the Guest Editorial in the September issue of *EHP*, Zoeller (2003) commented on an article by Lavado-Autric et al. (2003). Lavado-Autric et al. (2003) used the term "hypothyroxinemia" in this article to indicate that thyroxine (T_4) or free T_4 concentrations are low compared with values usually found at the same stage of pregnancy in normal women with adequate iodine intake, with or without the presence of clinical or subclinical hypothyroidism (when thyroid-stimulating hormone is above normal values). This is important because, in many instances, women in iodine-deficient populations are hypothyroxinemic; these women are not clinically hypothyroidal because they have normal or above-normal levels of circulating 3,5,3′-triiodothyronine (T_3) that can be supplied to the tissues.

It is important to note that the rats in the study were treated drastically by Lavado-Autric et al. (2003). The dams were first fed a diet with a low iodine content (LID) for 10 days and given an incredibly high amount of a goitrogen—1% perchlorate ($KClO_4$)—in the drinking water to lower the initial content of iodine-containing compounds in the thyroid gland; the 1% $KClO_4$ was then withdrawn. After dividing the rats into three groups, Lavado-Autric et al. (2003) treated one group with LID containing potassium iodide (LID-plus-KI) to ensure a normal iodine intake (approximately 10 μg iodine/day), the second group with LID alone (LID-1), and the third group with LID containing 0.005% $KClO_4$ (LID-2). This third treatment was used to further decrease thyroid uptake of the small amounts of iodine contained in the LID itself and in the supplements given to the rats throughout pregnancy and lactation to prevent nutritional deficiencies other than iodine.

In his editorial, Zoeller (2003) stated the following:

> Lavado-Autric et al. (2003) reported that subtle TH [thyroid hormone] insufficiency in the pregnant rat disrupts the migration of neurons in the fetal cortex and hippocampus.…

This was not subtle TH insufficiency. In fact, Lavado-Autric et al. (2003) stated that

> T_4 values in the LID-1 dams were well below normal (< 10% of the values of LID-plus-KI dams), and T_3 values remained normal. In LID-2 dams, however, T_3 values decreased, though much less markedly than T_4. Despite the decrease in T_3 values, the reproductive performance of these animals was normal, as was the postnatal growth of the pups at [postnatal day 40].

As a result,

> the subtle changes in cytoarchitectonic organization found in the progeny of both LID-1 and LID-2 dams indicate that the normal process of brain maturation … [is] likely to be impaired.

The author declares he has no competing financial interests.

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Zoeller RT. 2003. Thyroid toxicology and brain development: should we think differently? [Editorial]. Environ Health Perspect 111:A628.

Hypothyroxinemia: Zoeller's Response

Soldin addresses an important issue that was not fully developed in my editorial (Zoeller 2003a), specifically, the description of thyroid status in experimental animals designed to model human conditions. There are two separate issues in this regard. The first is that the language describing thyroid status is well defined for humans but not for experimental animals. Clinical assays for the various hormones of the pituitary–thyroid axis are standardized (and calibrated) across clinical chemistry laboratories, and reference ranges have been published for various subgroups of the population (e.g., Adams et al. 1995; Singh et al. 2003; Wiersinga 2001). Therefore, terms such as "hypothyroxinemia" and "subclinical (or mild) hypothyroidism" have very specific definitions for humans. In contrast, experimental studies are internally controlled (i.e., using control groups), and there are no reference ranges or assays that are calibrated across research laboratories performing these assays. Therefore, caution is needed when applying terms such as "subclinical hypothyroidism" to experimental animals.

The second, and more important, issue is that the vast majority of research focused on identifying the role of thyroid hormone (TH) in brain development has modeled severe hypothyroidism (reviewed by Schwartz 1983). Perhaps for this reason, the "clinical" symptoms of severe hypothyroidism in animals, including reduction in litter size, body weight, and brain size, and a delay in developmental landmarks such as tooth eruption and eye opening, have come to be viewed as cardinal developmental effects of TH insufficiency. Therefore, by association, if these "clinical" signs are not observed, the implication is that there would be no other effects on brain development. In large part, the work by Lavado-Autric et al. (2003) was testing whether "subtle" (my term) hypothyroidism could affect brain development (Zoeller 2003b).

By "subtle," I meant that maternal thyroxine (T_4) was reduced to a level below that of control animals but that overt effects on litter size, body weight, and other characteristics were not observed. Soldin is correct that the initial treatment of young adult female rats described by Lavado-Autric et al. (2003) was not subtle. Subgroups of these animals were treated for 10 days with a low iodine diet plus 1% potassium perchlorate in their drinking water. However, the animals were then taken off perchlorate treatment and placed on specifically designed diets for 3 months before being mated. Thus, the article by Lavado-Autric et al. is not about perchlorate treatment; it is about the sensitivity of the developing brain to TH insufficiency and the developmental timing of this vulnerability. The fact remains that there are no experimental studies designed to determine what might be considered a no effect level for maternal or neonatal TH insufficiency on brain development. However, this will be an important issue to clarify as we consider the significance of maternal hypothyroxinemia or the effects of thyroid toxicants on brain development.

The author declares he has no competing financial interests.

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Chemical Safety Requires Local Government Action

In "REACHing for Chemical Safety," Brown (2003) described the European Union's proposed Registration, Evaluation, and Authorisation of Chemicals (REACH) legislation and the shocking ineffectiveness of the laws currently governing toxic chemicals. Brown (2003) also revealed the depressing extent to which U.S. environmental officials, who are supposed to be advocates for the public and the environment, have instead fallen in line with the Bush administration's pro-business policies. Much-needed reform of U.S. policy on chemicals seems remote at best, but glimmers of hope, and possibly the future, exist outside the Washington, DC, Beltway. Last summer, San Francisco, California, became the first government jurisdiction in the United States to adopt the precautionary principle as a controlling environmental policy. San Francisco has also passed a resolution supporting a strong REACH in Europe, which would clearly benefit the people of California and elsewhere in the United States by promoting a safer global chemicals industry. Local communities and states can and must take environmental protection into their own hands; Californians are showing how bridges to Europe can help bypass the federal government altogether. Official San Francisco websites provide further information on the San Francisco ordinance on the Precautionary Principle Ordinance (SF Environment 2003) and the REACH Resolution (City and County of San Francisco Board of Supervisors 2003).

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CORRECTIONS

The November 2003 Forum article "New Data on Methylmercury and Fetuses" [Environ Health Perspect 111:A753 (2003)] incorrectly stated, in reference to methylmercury poisoning at Minamata Bay in the mid-1950s, "The hair mercury of pregnant women in Minamata ranged from 25 to 50 ppm." In fact, there are no direct data on the hair mercury concentrations of the pregnant Minamata women whose children had health problems associated with methylmercury poisoning. *EHP* regrets the error.

In Tables 3–7 of "Assessing Potential Risk of Heavy Metal Exposure from Consumption of Home-Produced Vegetables by Urban Populations" by Hough et al. [Environ Health Perspect 112:215–221 (2004)], the values "× 10" should be "× 100." In Table 3, the β_1 values for lettuce should be -3.59×10^{-1} and the β_1 value for cabbage should be -6.23×10^{-2}.

A footnote was omitted from the body of Tables 2–4 in the article "Urinary Levels of Seven Phthalate Metabolites in the U.S. Population from the National Health and Nutrition Examination Survey (NHANES) 1999–2000" by Silva et al. [Environ Health Perspect 112:331–338 (2004)]. Footnote "*a*," indicating the meaning of italics in each of these tables, should have been placed in the second line under "GM (geometric mean). *EHP* apologizes for the error. Also, Silva et al. would like to clarify that the "No. (%)" values in Tables 2–4 indicate sample size (percentage of detection).

Orlando et al. discovered an error in their article, "Endocrine-Disrupting Effects of Cattle Feedlot Effluent on an Aquatic Sentinel Species, the Fathead Minnow" [Environ Health Perspect 112:353–358 (2004)]. In Table 3, a zero was omitted from the value for gonadal mass under "Intermediate Site"; the correct value is 0.088 ± 0.01 instead of 0.88 ± 0.01.